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an Bine (File 481)
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RELOADED
***Kompa Middle Eat/Africa/Mediterranean (File 585)
***Kompa Aia/Pacific (File 592)
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***Kompa Canada (File 594)
***CANCERLIT (File 159)
***Information Science Abtract (File 202)

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File   1:ERIC  1966-2001/Sep 06
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      Set  Items  Description
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? set hi ;set hi

       27sep01 14:10:41 User208760 Session D1941.1
            $0.55    0.156 DialUnits File1
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     $0.60  Estimated total session cost   0.156 DialUnits

File 410:Chronolog(R)  1981-2001/Sep
       (c) 2001 The Dialog Corporation

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PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
? begin 5,73,155,399

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     $0.40  TYMNET
     $0.40  Estimated cost this search
     $1.00  Estimated total session cost   0.223 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File   5:Biosis Previews(R)  1969-2001/Sep W4
         (c) 2001 BIOSIS
  File  73:EMBASE  1974-2001/Sep W4
         (c) 2001 Elsevier Science B.V.
*File  73: For information about Explode feature please
see Help News73.
  File 155:MEDLINE(R)  1966-2001/Oct W3
  File 399:CA SEARCH(R)  1967-2001/UD=13514
         (c) 2001 AMERICAN CHEMICAL SOCIETY
*File 399: Use is subject to the terms of your user/customer agreement.
RANK charge added; see HELP RATES 399.

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? s nip145

      S1       0  NIP145
? s nip45

      S2      17  NIP45
? rd s2

...completed examining records
      S3       8  RD S2 (unique items)
? t s3/7/all


 3/7/1     (Item 1 from file: 5)
DIALOG(R)File   5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.

13147731   BIOSIS NO.: 200100354880
Tumor necrosis factor receptor-associated factor (TRAF)2 represses the T
  helper cell type 2 response through interaction with NFAT-interacting
  protein (NIP45).
AUTHOR: Lieberson Rebecca; Mowen Kerri A; McBride Kathryn D; Leautaud
  Veronica; Zhang Xiankui; Suh Woong-Kyung; Wu Lin; Glimcher Laurie H(a)
AUTHOR ADDRESS: (a)Department of Immunology and Infectious Diseases,
  Harvard School of Public Health, 651 Huntington Ave., Boston, MA, 02115:
  lglimche@hsph.harvard.edu**USA
JOURNAL: Journal of Experimental Medicine  194 (1):p89-98 July 2, 2001
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: Recently we have identified a novel protein NIP45 (nuclear
  factor of activated T cells (NFAT)-interacting protein) which
  substantially augments interleukin (IL)-4 gene transcription. The
  provision of NIP45 together with NFAT and the T helper cell type 2
  (Th2)-specific transcription factor c-Maf to cells normally refractory to
  IL-4 production, such as B cells or Th1 clones, results in substantial
  IL-4 secretion to levels that approximate those produced by primary Th2
  cells. In studies designed to further our understanding of NIP45
  activity, we have uncovered a novel facet of IL-4 gene regulation. We
  present evidence that members of the tumor necrosis factor
  receptor-associated factor (TRAF) family of proteins, generally known to
  function as adapter proteins that transduce signals from the tumor
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necrosis factor receptor superfamily, contribute to the repression of IL-4 gene transcription and that this effect is mediated through their interaction with **NIP45**.

3/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

12944244 BIOSIS NO.: 200100151393
NF-AT-interacting protein **NIP45** and methods of use therefor.
AUTHOR: Glimcher Laurie H(a); Hodge Martin R
AUTHOR ADDRESS: (a)West Newton, MA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1236 (3):pNo Pagination July 18, 2000
MEDIUM: e-file
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Isolated nucleic acid molecules encoding a novel protein, **NIP45**, that interacts with members of the Nuclear Factor of Activated T cell (NF-AT) family of proteins, are disclosed. The invention further provides antisense nucleic acid molecules, recombinant expression vectors containing a nucleic acid molecule of the invention, host cells into which the expression vectors have been introduced and non-human transgenic animals carrying a **NIP45** transgene. The invention further provides isolated **NIP45** proteins and peptides, **NIP45** fusion proteins and anti-**NIP45** antibodies. Methods of using the **NIP45** compositions of the invention are also disclosed, including methods for detecting **NIP45** protein or mRNA in a biological sample, methods of modulating **NIP45** activity in a cell, and methods for identifying agents that modulate an interaction between **NIP45** and an NF-AT family protein.

3/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

10714730 BIOSIS NO.: 199799335875
NF-AT-driven interleukin-4 transcription potentiated by **NIP45**.
AUTHOR: Hodge Martin R(a); Chun Hyung J(a); Rengarajan Jyothi; Alt Aya; Lieberson Rebecca; Glimcher Laurie H
AUTHOR ADDRESS: (a)Millennium Pharm. Inc., Cambridge, MA 02139**USA
JOURNAL: Science (Washington D C) 274 (5294):p1903-1905 1996
ISSN: 0036-8075
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The induction of cytokine gene transcription is mediated in part by the nuclear factor of activated T cells (NF-AT). Factors involved in the mechanisms of NF-AT-mediated transcription are not well understood. A nuclear factor that interacted with the Rel homology domain (RHD) of NF-ATp was identified with the use of a two-hybrid interaction trap. Designated **NIP45** (NF-AT interacting protein), it has minimal similarity to any known genes. Transcripts encoding this factor were enriched in lymphoid tissues and testes. **NIP45** synergized with NF-ATp and the proto-oncogene c-Maf to activate the interleukin-4 (Il-4) cytokine promoter; transient overexpression of **NIP45** with NF-ATp and c-maf in B lymphoma cells induced measurable endogenous IL-4 protein production. The identification of **NIP45** advances our understanding of gene activation of cytokines, critical members of the immune response.

3/7/4 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

10910020 EMBASE No: 2000404628
Reconstitution of T cell-specific transcription directed by composite NFAT/Oct elements
Bert A.G.; Burrows J.; Hawwari A.; Vadas M.A.; Cockerill P.N.
Dr. P.N. Cockerill, Hanson Center for Cancer Research, Inst. for Med./Veterinary Science, Rundle Mall Post Office, P.O. Box 14, Adelaide, SA 5000 Australia
AUTHOR EMAIL: peter.cockerill@imvs.sa.gov.au
Journal of Immunology (J. IMMUNOL.) (United States) 15 NOV 2000, 165/10 (5646-5655)
CODEN: JOIMA ISSN: 0022-1767
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 60

The complex nature of most promoters and enhancers makes it difficult to identify key determinants of tissue-specific gene expression. Furthermore, most tissue-specific genes are regulated by transcription factors that have expression profiles more widespread than the genes they control. NFAT is an example of a widely expressed transcription factor that contributes to several distinct patterns of cytokine gene expression within the immune system and where its role in directing specificity remains undefined. To investigate distinct combinatorial mechanisms employed by NFAT to regulate tissue-specific transcription, we examined a composite NFAT/AP-1 element from the widely active GM-CSF enhancer and a composite NFAT/Oct element from the T cell-specific IL-3 enhancer. The NFAT/AP-1 element was active in the numerous cell types that express NFAT, but NFAT/Oct enhancer activity was T cell specific even though Oct-1 is ubiquitous. Conversion of the single Oct site in the IL-3 enhancer to an AP-1 enabled activation outside of the T cell lineage. By reconstituting the activities of both the IL-3 enhancer and its NFAT/Oct element in a variety of cell types, we demonstrated that their T cell-specific activation required the lymphoid cofactors **NIP45** and OCA-B in addition to NFAT and Oct family proteins. Furthermore, the Oct family protein Brn-2, which cannot recruit OCA-B, repressed NFAT/Oct enhancer activity. Significantly, the two patterns of combinatorial regulation identified in this study mirror the cell-type specificities of the cytokine genes that they govern. We have thus established that simple composite transcription factor binding sites can indeed establish highly specific patterns of gene expression.

3/7/5 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10555153 20216392 PMID: 10755616
Sequential involvement of NFAT and Egr transcription factors in FasL regulation.
Rengarajan J; Mittelstadt PR; Mages HW; Gerth AJ; Kroczek RA; Ashwell JD; Glimcher LH
Department of Immunology and Infectious Diseases, Harvard School of Public Health, Boston, Massachusetts 02115, USA.
Immunity (UNITED STATES) Mar 2000, 12 (3) p293-300, ISSN 1074-7613 Journal Code: CCF
Contract/Grant No.: AG37833, AG, NIA; AI98005, AI, NIAID
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The critical function of NFAT proteins in maintaining lymphoid homeostasis was revealed in mice lacking both NFATp and NFAT4 (DKO). DKO mice exhibit increased lymphoproliferation, decreased activation-induced cell death, and impaired induction of FasL. The transcription factors Egr2 and Egr3 are potent activators of FasL expression. Here we find that Egr2

and Egr3 are NFAT target genes. Activation of FasL occurs via the NFAT-dependent induction of Egr3, as demonstrated by the ability of exogenously provided NFATp to restore Egr-dependent FasL promoter activity in DKO lymph node cells. Further, Egr3 expression is enriched in Th1 cells, suggesting a molecular basis for the known preferential expression of FasL in the Th1 versus Th2 subset.
Record Date Created: 20000426

3/7/6 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

130321592 CA: 130(24)321592t PATENT
Cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
INVENTOR(AUTHOR): Zhou, Hong; Zhao, Jiiuquiq; Liu, Derong
LOCATION: UK,
ASSIGNEE: Zeneca Limited
PATENT: PCT International ; WO 9921993 A1 DATE: 19990506
APPLICATION: WO 98GB3141 (19981021) *GB 9722388 (19971024)
PAGES: 84 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/12A; C07K-014/47B; C07K-016/18B; C12N-015/11B; G01N-033/68B; C12Q-001/68B
DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CU; CZ; DE; DK; EE; ES; FI; GB; GE; GH; GM; HR; HU; ID; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR; TT; UA; UG; UZ; VN; YU; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; SD; SZ; UG; ZW; AT; BE; CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG
SECTION:
CA203003 Biochemical Genetics
CA206XXX General Biochemistry
CA213XXX Mammalian Biochemistry
CA263XXX Pharmaceuticals
IDENTIFIERS: NIP45 interleukin 4 gene transcription factor human, sequence NIP45 cDNA human
DESCRIPTORS:
Nucleic acids...
antisense; cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
Antibodies... Drug screening... Interleukin 4... Molecular cloning...
cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
Primers(nucleic acid)...
for diagnostic identification; cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
cDNA sequences...
for human NIP-45 interleukin-4 gene transcriptional trans-activator
Transcription factors...
NFAT-1 (nuclear factor, activated T-cell, 1); cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
Transcription factors...
NIP-45 (interleukin 4 gene-activating); cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
Protein sequences...
of human NIP-45 interleukin-4 gene transcriptional trans-activator
Molecular diagnosis...
PCR primers for; cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
CAS REGISTRY NUMBERS:
223698-49-5 amino acid sequence; cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator
223698-50-8 223698-51-9 nucleotide sequence; cloning and cDNA sequence of human NIP-45 interleukin-4 gene transcriptional trans-activator

3/7/7 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

128021847 CA: 128(3)21847h PATENT
Methods and compositions for regulating T cell subsets by modulating transcription factor activity
INVENTOR(AUTHOR): Glimcher, Laurie H.; Hodge, Martin R.; Ho, I-Cheng
LOCATION: USA
ASSIGNEE: President and Fellows of Harvard College; Glimcher, Laurie H.; Hodge, Martin R.; Ho, I-Cheng
PATENT: PCT International ; WO 9739721 A2 DATE: 19971030
APPLICATION: WO 97US6708 (19970423) *US 636602 (19960423) *US 755584 (19961125) *US 755592 (19961125)
PAGES: 151 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-000/A
DESIGNATED COUNTRIES: AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; CA; CH; CN; CZ; DE; DK; EE; ES; FI; GB; GE; GH; HU; IL; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG; MK; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; TJ; TM; TR; TT; UA; UG; US; US; US; UZ; VN; YU; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; KE; LS; MW; SD; SZ; UG; AT ; BE; CH; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE; SN; TD; TG
SECTION:
CA215001 Immunochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: Th2 cytokine transcription factor modulator antibody
DESCRIPTORS:
Transcription factors...
c-maf; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Genes(animal)...
for Th2-assocd. cytokine; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Transcription factors...
maf; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Allergies... Autoimmune diseases... B cell(lymphocyte)... cDNA sequences... DNA... Hematopoietic stem cell... Immunomodulators... Infection... Interleukin 4... Lymphocyte... Protein sequences... Reporter genes... T cell(lymphocyte)... Th1 cell... Th2 cell... Transcription factor AP-1... Transgenes... Transplant(organ)... Tumors(animal)... Yeast...
methods and compns. for regulating T cell subsets by modulating transcription factor activity
Transcription factors...
NF-AT or nuclear factor-activated T cell; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Antisense DNA... Fusion proteins(chimeric proteins)... Nucleic acids...
Transcription factors...
NIP45; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Animal...
nonhuman transgenic; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Animal cells...
nonlymphoid; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Transcription factors...
p18; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Transcription factors...
small maf; methods and compns. for regulating T cell subsets by modulating transcription factor activity
Cytokines...
Th2-assocd.; methods and compns. for regulating T cell subsets by modulating transcription factor activity

7/7/6 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

10965234 EMBASE No: 2001009739
Altering the Th1/Th2 balance as a **therapeutic** strategy in asthmatic diseases
Ray A.; Cohn L.
A. Ray, Department of Internal Medicine, Yale University, School of Medicine, 333 Cedar Street, New Haven, CT 06520-8057 United States
AUTHOR EMAIL: Anuradha.Ray@yale.edu
Current Opinion in Investigational Drugs (CURR. OPIN. INVEST. DRUGS) (United Kingdom) 2000, 1/4 (442-448)
CODEN: CIDRE ISSN: 0967-8298
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 81

The identification of distinct T helper (Th)-cell subsets that differ in cytokine production and effector functions not only provides a framework for normal immune responses to infection and injury, but also for many disease processes. Studies in both humans and animals indicate that airway inflammation in allergic asthma is orchestrated by CD4+ Th2-cells that secrete the cytokines IL-4, IL-5 and IL-13. Many studies also suggest that IFNgamma, secreted by Th1-cells, suppresses the development and effector functions of Th2-cells. Cross-regulation of Th1/Th2 responses has been demonstrated in many experimental systems including models of allergic inflammation/asthma. A challenging concept that has evolved as a result is the use of **therapeutic** modalities that will modulate the Th1/Th2 balance in asthma without deleterious side effects. In the clinical trial arena, the unmet challenging goal remains to convert the concept of Th1/Th2 balance modulation, without deleterious side effects, into clinical practice for the management of asthmatic disease.

7/7/12 (Item 12 from file: 73)
DIALOG(R)File 73:EMBASE

07473816 EMBASE No: 1998404012
Nuclear factor kappa B: A pivotal role in the systemic inflammatory response syndrome and new target for **therapy**
Christman J.W.; Lancaster L.H.; Blackwell T.S.
J.W. Christman, Department of Medicine, T1217 Medical Center North, Vanderbilt Univ. School of Medicine, Nashville, TN 37322-2650 United States
AUTHOR EMAIL: john.christman@mcmail.vanderbilt.edu
Intensive Care Medicine (INTENSIVE CARE MED.) (Germany) 1998, 24/11 (1131-1138)
CODEN: ICMED ISSN: 0342-4642
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 81

NF-kappaB is an important transcription factor complex that appears to play a fundamental role in regulating acute inflammation through activation of the cytokine cascade and production of other pro-inflammatory mediators. There is increasing evidence that NF-kappaB is important in the pathobiology of disease states such as SIRS, MODS and ARDS; therefore,

therapeutic interventions aimed at limiting NF-kappaB activation and down-regulating production of inflammatory mediators could prove to be beneficial in decreasing host-derived tissue injury and organ dysfunction. Specific interventions that hold promise for suppressing NF-kappaB activation include the use of antioxidants, inhibition of NIK and the IKK signalsome, **treatment** with proteasome inhibitors, induction of endotoxin tolerance and, possibly the use of corticosteroids in selected patients.

7/7/17 (Item 17 from file: 73)
DIALOG(R)File 73:EMBASE

06451957 EMBASE No: 1996114901
Targeting signal transduction for disease **therapy**
Levitski A.
Department of Biological Chemistry, Institute of Life Sciences, The Hebrew University of Jerusalem,Givat Ram, Jerusalem 91904 Israel
Current Opinion in Cell Biology (CURR. OPIN. CELL BIOL.) (United Kingdom) 1996, 8/2 (239-244)
CODEN: COCBE ISSN: 0955-0674
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

With the advance in the molecular understanding of disease processes, it has been appreciated that many diseases result from the malfunctions of signaling pathways. This recognition has led to intensive research and the development of **therapies** based on the interception of cellular signaling in diseased cells. In the past two years, success has been achieved using a blocker of the farnesylation of Ras as a tumor inhibitor, a JAK-2 blocker as an efficient inhibitor of recurrent pre-B cell acute lymphoblastic leukemia, and a platelet-derived growth factor receptor kinase as a blocker of restenosis.